



Trigger Finger Treatment

Tratamento do Dedo em Gatilho

João Carlos Belloti¹  Edson Sasahara Sato¹  Flavio Faloppa¹ 

¹ Department of Orthopedics and Traumatology, Escola Paulista de Medicina, Universidade Federal de São Paulo, São Paulo, SP, Brazil

Address for correspondence Edson Sasahara Sato, PhD, Department of Orthopedics and Traumatology, Universidade Federal de São Paulo, Escola Paulista de Medicina, Rua Borges Lagoa, 786, São Paulo, SP, 04038-001, Brazil (e-mail: edsonsasahara@gmail.com).

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Abstract

Trigger finger is a frequent condition. Although tenosynovitis and the alteration of pulley A1 are identified as triggering factors, there is no consensus on the true cause in the literature, and its true etiology remains unknown. The diagnosis is purely clinical most of the time. It depends solely on the existence of finger locking during active bending movement. Trigger finger treatment usually begins with nonsurgical interventions that are instituted for at least 3 months. In patients with initial presentation with flexion deformity or inability to flex the finger, there may be earlier indication of surgical treatment due to pain intensity and functional disability.

Keywords

- ▶ trigger finger/ diagnostic
- ▶ trigger finger/therapy
- ▶ trigger finger/surgery
- ▶ tenosynovitis

In the present review article, we will present the modalities and our algorithm for the treatment of trigger finger.

Resumo

O dedo em gatilho é uma afecção frequente. Não obstante a tenossinovite e a alteração da polia A1 serem identificados como fatores desencadeantes, não há consenso sobre a verdadeira causa na literatura, sendo que a sua verdadeira etiologia permanece desconhecida. O diagnóstico é puramente clínico na maior parte das vezes. Ele depende unicamente da existência do travamento do dedo no decorrer da movimentação flexão ativa. O tratamento do dedo em gatilho geralmente se inicia com intervenções não cirúrgicas que são instituídas por pelo menos 3 meses. Nos pacientes em quem haja apresentação inicial com deformidade em flexão ou incapacidade de flexão do dedo, pode haver indicação mais precoce do tratamento cirúrgico em razão da intensidade do quadro algico e da incapacidade funcional do paciente.

Palavras-chave

- ▶ dedo em gatilho/ diagnóstico
- ▶ dedo em gatilho/ terapia
- ▶ dedo em gatilho/ cirurgia
- ▶ tenossinovite

No presente artigo de revisão, apresentaremos as modalidades e o nosso algoritmo para o tratamento do dedo em gatilho.

Introduction

Notta¹ was the first to describe trigger finger as a pathology triggered by changes in the flexor tendon and its sheath. Hueston et al.² demonstrated in a study that the spiral arrangement in

the architecture of intratendinous fibers leads to the formation of a nodulation when passing through a stenosis point.

Brozovich et al.³ described histological changes in the A1 pulley that undergoes metaplasia with fibroblast replacement by chondrocytes being one of the reasons for increased

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pressure between the tendon and the osteofibrous tunnel in trigger finger.

Although tenosynovitis and the alteration of the A1 pulley are identified as triggering factors, there is no consensus on the true cause in the literature, and its true etiology remains unknown.⁴

The definition of trigger finger is that it is a condition in which the flexor tendon has its sliding blocked when passing in the pulley system, notably on the A1 pulley, not being able to tour and return to the initial position, preventing the finger from making its natural bending and extension movement.

This condition affects 2 to 3% of the population, more in the 6th decade of life.⁵ The most affected finger is the annular, followed by the middle finger, thumb, minimum and index fingers. The dominant hand is affected 70% of the time. Women have twice the chance of having trigger finger than men.⁶

Patients with diabetes are 15% more likely to develop trigger finger than the general population. However, when treated by surgery, it has the same evolution as in nondiabetic patients, as well as not having a greater chance of presenting multiple trigger fingers.⁷

Sometimes, trigger finger has been associated with carpal tunnel release surgery. Corroborating this conception, in a systematic review of the literature, Lin et al.⁸ described an average rate of 8.5% of disease development after the procedure for the opening of the flexor retinaculum.

Trigger finger also affects children being commonly referred to as a "congenital trigger", with differences in clinical presentation.

The "congenital trigger" in the thumb is 10 times more common than that in the other fingers, and patients usually present it at ~ 2 years old.⁹ The incidence of "congenital trigger" is 3.3 per 1,000 live births.¹⁰ Although the etiology of the "congenital trigger" is unknown, it is known that it is an acquired condition. Authors evaluated 7,000 newborns and found no trigger fingers.¹¹

Clinical presentation

The symptom usually starts with pain in the area coincident with the A1 pulley in the fingers. The occurrence arises with no apparent reason or after a continued manual preambular action. Pain can continue for months without change in mobility, or progress to a blockage of finger movement, initially in the morning, persisting in this situation or advancing to a more constant trigger, at all active finger flexions. Similarly, the trigger phenomenon can have a sudden onset without a painful prodrome. This manifestation merely arrives in a digit or in more than one at the same time.

Quinnell⁴ studied the results of hydrocortisone injection into the A1 pulley in 48 trigger fingers of 43 patients. He divided the different groups of trigger fingers into five types, relating them to flexion and extension, being type "0" with normal movement, type "I" the sporadic trigger, type "II" the actively correctable trigger, type "III" the only passively correctable trigger, and type "IV" with fixed deformity. This

Table 1 Trigger finger rating according to Green DP (Green DP, personal communication, 1997)¹²

Type	Clinical feature
I (pretrigger)	Pain; history of locking, but not demonstrable on clinical examination; sensitivity to palpation above the A1 pulley
II (active)	Demonstrable locking, but the patient is able to actively extend the finger
III (passive)	Demonstrable locking requiring passive extension (grade IIIA) or impossibility of active finger flexion (grade IIIB)
IV (contracture)	Demonstrable locking with a flexion contracture of the proximal interphalangeal joint

classification was modified by Green DP (Green DP, personal communication, 1997)¹² according to **Table 1**.

It is interesting to note that trigger finger is a phenomenon that occurs only in the course of active finger flexion.

Finger unlocking may result from active movement or require an external force for this purpose.

Fortuitously, the finger will be permanently flexed with both active and passive impediment in the extension, having the unlock inexecutable, regardless of the energy used. Other times, it will be extended perennially, having impediment in active flexion.

Sometimes, the proximal interphalangeal joint progresses to a flexion contracture, usually correctable by passive extension, despite pain related to finger rectification.

Moreover, trigger finger may be correlated with deposit diseases (sarcoidosis, amyloidosis), diseases with altered metabolism (hypothyroidism, diabetes), as well as autoimmune diseases (rheumatoid arthritis, lupus).¹³

Diagnostic

The diagnosis is clinical most of the time. It depends solely on the existence of finger locking during active movement.

If the finger is permanently extended or flexed, having been unable to complete the active flexion or passive extension, respectively in this order, the determination of the diagnosis will be more intricate.

In the extended digit with impracticality in active flexion, it is imperative to rule out a closed injury of the flexor tendons. In this situation, after stabilization of the middle phalanx, the patient will usually be able to actively flex the distal phalanx, proving its integrity. Another possibility to be differentiated will be that of contracture in extension due to joint damage, and radiography is interesting in this situation.

On a persistently flexed finger, with incapacity in passive extension, it will be essential to differentiate from a tendon adhesion of the flexor tendons or from the contracture in flexion of the finger.

In case of uncertainty, the ultrasound examination will show the flexor tendon, its morphological changes, as well as the thickening of the flexor pulley.¹⁴

Treatment

Trigger finger treatment usually begins with nonsurgical interventions that are instituted for at least 3 months. In patients with initial presentation of flexion deformity or inability to flex the finger, there may be earlier indication of surgical treatment due to pain intensity and functional disability.

Non-surgical methods

Physiotherapy

Salim et al.,¹⁵ in a study comparing the efficacy of infiltration with corticosteroid versus physiotherapy, physiotherapy achieved 68.6% success versus 97% of infiltration in the treatment of trigger finger.

In one study by Watanabe et al.,¹⁶ physiotherapy performed by the mothers of the patients with “congenital trigger thumb” themselves resulted in the cure rate of 80% for stage 2 (trigger that unlocks in active movement) and of 25% for stage 3 (trigger that unlocks in passive movement only).

Immobilization

Immobilization was described by Patel et al.¹⁷ by means of an orthosis that maintained the metacarpophalangeal joint at 10° to 15° flexion with free interphalangeal joints for an average time of 6 weeks. Treatment on the index fingers, mean annular, and minimum was successful in 66%, and in thumbs, in 50%.

Infiltration

Corticosteroid infiltration is the most commonly used nonsurgical treatment modality, and has been described by numerous authors.^{18–22}

Sato et al.,¹⁸ in a prospective randomized study comparing the results of trigger finger treatment by infiltration versus percutaneous release versus open surgery, obtained cure in 86% with methylprednisolone injection.

Mardani-Kivi et al.¹⁹ compared the infiltration results with and without the aid of ultrasound, having 94% success also in both groups.

Despite the encouraging results with glucocorticoids, caution is needed in diabetic patients who may have significant increases in glycemic levels.²⁰

Roberts et al.²¹ compared the results of infiltration with triamcinolone, dexamethasone, methylprednisolone. They concluded that trigger fingers treated with triamcinolone are more likely to require a second injection than those treated with methylprednisolone or dexamethasone.

Newport et al.,²² in a retrospective study, analyzed the safety and efficacy of the treatment of trigger finger, initially through the infiltration of betamethasone. They examined 235 patients with 338 trigger fingers, 71% in the right hand, 63% women and 37% men. The mean follow-up period was of 35 months. As for the results, 49% improved after infiltration, 23% after 2 infiltrations, and 5% after 3 infiltrations. The rest of the patients who did not improve, totaling 33%, underwent



Fig. 1 Corticosteroid injection inside the osteofibrous tunnel of the A1 pulley.

open surgical treatment through the surgical release of the A1 pulley. According to these authors, the initial treatment of trigger finger should be with corticosteroid infiltration, and its prior use would not harm the outcome of an operative treatment if necessary.

Our preference is for up to 2 infiltrations with an interval of at least 1 month, using methylprednisolone acetate or betamethasone dipropionate, inside the osteofibrous tunnel.

Technical

We perform a local anesthesia with 2 mL of 0.5% subcutaneous lidocaine at the site coincident with the A1 pulley of the affected finger. Approximately after 5 minutes, we insert the needle into the flexor tendon through the A1 pulley. We retract the needle until we feel decreased resistance in the syringe embolus and then inject the corticosteroid. We prefer to use methylprednisolone acetate or betamethasone dipropionate (→ **Figure 1**).

Surgical Methods

Surgical therapy is usually indicated when nonsurgical treatment fails.

Surgery consists of the incision of the A1 pulley. It can be performed in the traditional open form or percutaneously.

Percutaneous method

Eastwood et al.²³ were the pioneers in percutaneous release, presenting 94% satisfactory results.

Sato et al.²⁴ performed percutaneous release of trigger finger with 96% remission, with recurrence in Quinnell type I patients. They concluded that the sporadic trigger (type I), which does not occur at all finger flexions, would not be indicated for percutaneous release, since the extinction of the lock is indicative of the success of the surgery.

In a second study, Sato et al.¹⁸ excluded the type I trigger, having 100% cure with percutaneous release.

Bain et al.²⁵ found that the neurovascular bundle of the thumb is located 2 mm from the A1 pulley, becoming more vulnerable to needle injury. Despite this, Sato et al.¹⁸ performed percutaneous release on the thumb and had no lesion of digital nerves.

Because it is a closed method, authors such as Lee et al.²⁶ compared percutaneous release with and without ultrasound aid and concluded that visualization of the A1 pulley by imaging examination decreases the possibility of incomplete incisions.

Technique

The patient is positioned by keeping the affected hand in supination. With a skin marker pen, we make a stroke on the longitudinal axis of the finger. Two milliliters of local anesthetic (2% lidocaine, without vasoconstrictor) are infiltrated in the subcutaneous tissue and around the A1 pulley (►Figure 2a). The interphalangeal aniles and metacarpophalangeal joints of the fingers should be in full extension in order to dorsally displace the neurovascular bundles, reducing the possibility of damaging these structures during the procedure. The bezel of the needle should be positioned towards its cut parallel to the longitudinal axis of the finger (►Figure 2b). A 40 × 12 hypodermic needle is inserted perpendicular to the skin at the site corresponding to the A1 pulley. It confirms the positioning of the needle in the tendon, flexing the finger and observing the concomitant displacement of the needle (►Figure 2c). Then, the needle is retracted until the concomitant oscillation of the needle stops with the passive movement of the finger. Longitudinal movements are performed in the direction of the pulley in order to section it (►Figure 2d). The patient is asked to perform active flex-

ion movements of the operated finger to confirm if there was total release of the pulley. If necessary, the longitudinal movements are repeated with the needle until the complete release of the trigger is verified. Then, the needle is removed, finishing the procedure, and the dressing is performed, without the need for immobilization. Patients are instructed to avoid manual activities and to perform cryotherapy with a bag containing ice cubes on the day of the procedure.

Open method

Open surgery is the most classic surgical method and is usually evidenced as a method with high rates of satisfactory results.

Turowski et al.²⁷ showed satisfactory results with open surgery of 97%.

Leung et al.²⁸ performed the open release of 161 thumbs with "congenital trigger", obtaining 95% satisfactory results.

Technique

Patient in supine position after anesthesia. A transverse cutaneous incision is made at the site corresponding to the flexor pulley. The digital nerves on each side are identified and gently removed, and the flexor pulley is visualized. (►Figure 3a) The pulley is incised in its longitudinal direction and the tendon is exposed (►Figure 3b-c). After suturing the skin, the wound is covered with sterile dressing, without immobilization, for ~ 1 week.

Final Considerations

Amirfeiz et al.²⁹ conducted a systematic review comparing trigger finger treatments in adults. They concluded that corticosteroid infiltration as the first treatment is a reasonable option. Percutaneous release performed by a trained person is safe. In the failure of infiltration, surgical methods are indicated.

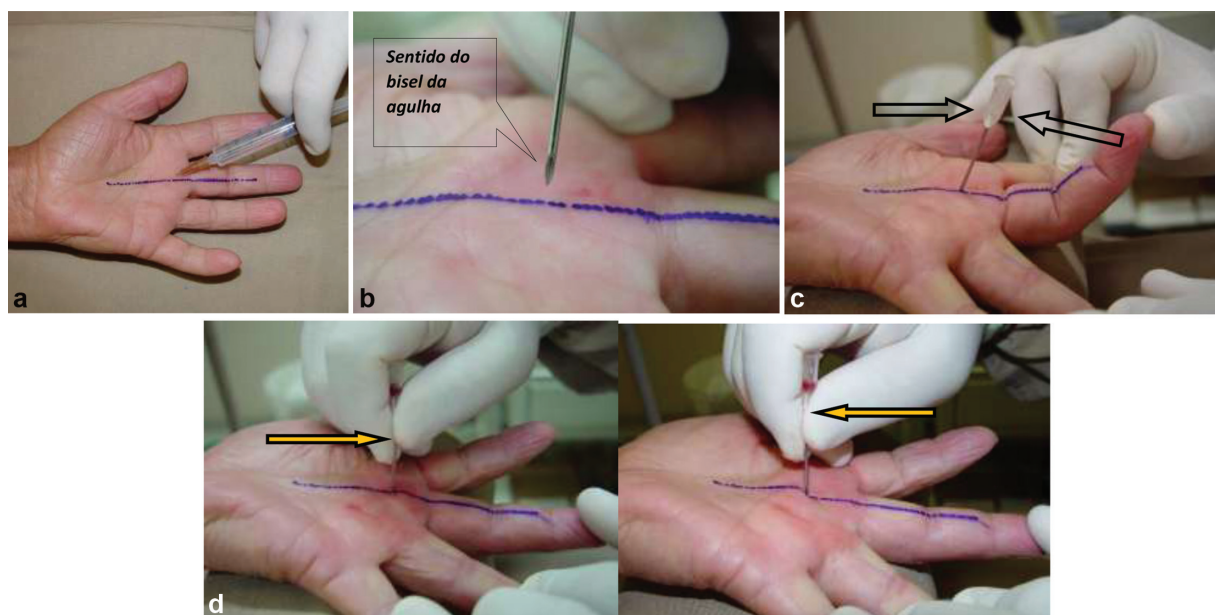


Fig. 2 (a) Anesthesia of the skin and subcutaneous cellular tissue, with the longitudinal axis of the finger demarcated. (b) Correct positioning of the needle bezel at the time of its introduction. (c) Displacement of the needle when passively flexing the finger. (d) Sectioning pulley A1.

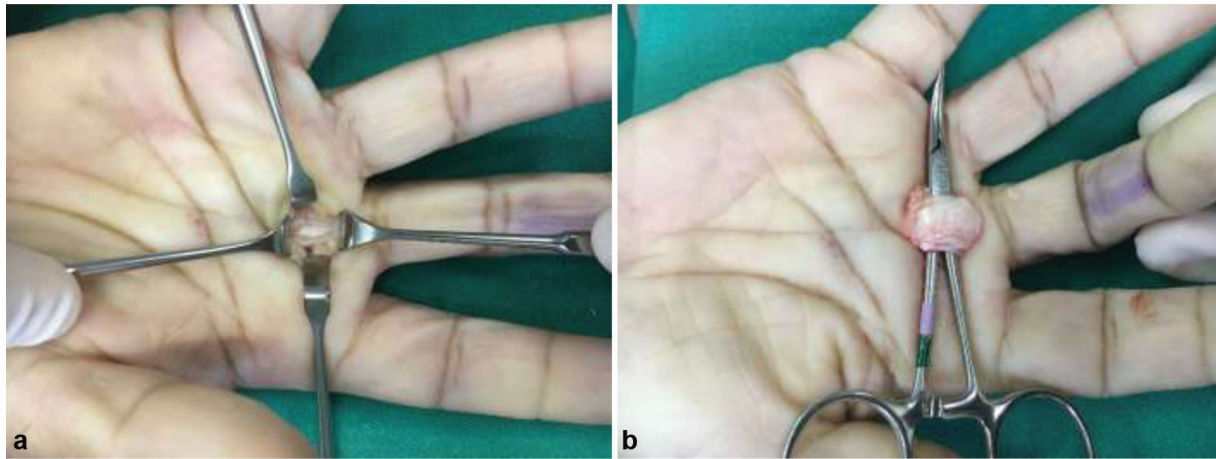


Fig. 3 (a) Incision and opening of the flexor pulley. (b) Exposed tendon.

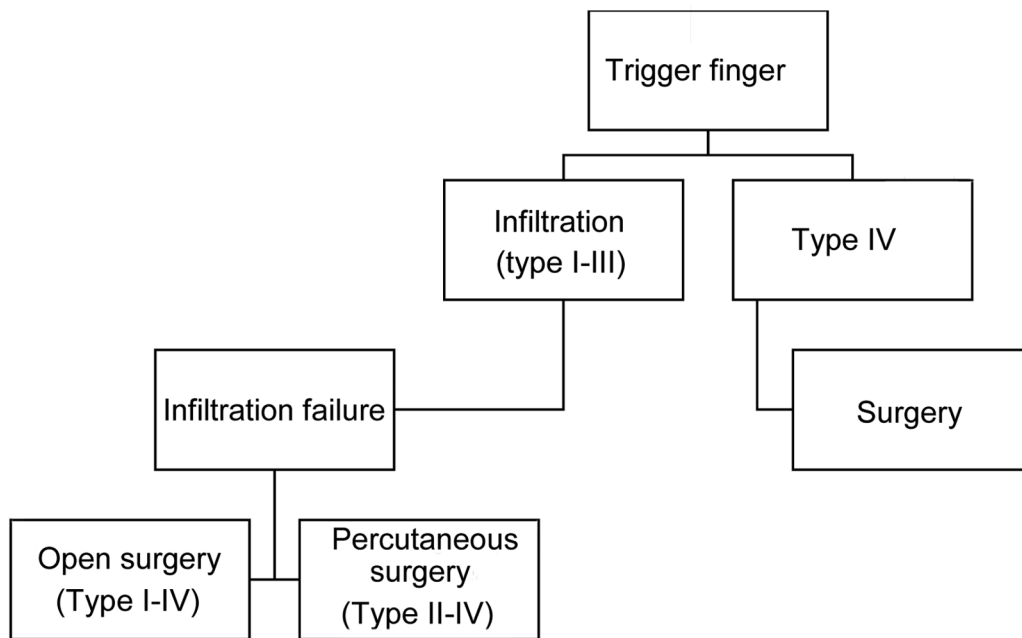


Fig. 4 Trigger finger treatment algorithm.

We agree that infiltration should be indicated as the first therapeutic option; however, in diabetic patients, who may have their blood glucose elevated considerably, we must be cautious, especially in patients with glycemic levels not counterbalanced by medication.

Fiorini et al.³⁰ conducted a systematic review of trigger finger treatment and concluded that open surgery has a lower recurrence rate when compared with patients treated with infiltration.

Brozovich et al.³ pointed out that from a purely financial point of view, women without diabetes with trigger thumb should receive up to two infiltrations before being submitted to percutaneous release.

We believe that although surgical methods (open and percutaneous) have higher cure rates when compared with infiltration, as our study¹⁸ showed, they are more expensive options, and should be reserved for failure of nonsurgical treatment.

We present below our treatment algorithm (► **Figure 4**).

Conflict of Interests

The authors have no conflict of interests to declare.

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